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Polycyclic aromatic hydrocarbon exposure, obesity and childhood asthma in an urban cohort

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ABSTRACT

Background: Exposure to traffic-related air pollutants, including polycyclic aromatic hydrocarbons (PAHs) from traffic emissions and other combustion sources, and childhood obesity, have been implicated as risk factors for developing asthma. However, the interaction between these two on asthma among young urban children has not been studied previously.

Methods: Exposure to early childhood PAHs was measured by two week residential indoor monitoring at age 5–6 years in the Columbia Center for Children's Environmental Health birth cohort ($n=311$). Semivolatile [e.g., methylphenanthrenes] and nonvolatile [e.g., benzo(a)pyrene] PAHs were monitored. Obesity at age 5 was defined as a body mass index (BMI) greater than or equal to the 95th percentile of the year 2000 age- and sex-specific growth charts (Center for Disease Control). Current asthma and recent wheeze at ages 5 and 7 were determined by validated questionnaires. Data were analyzed using a modified Poisson regression in generalized estimating equations (GEE) to estimate relative risks (RR), after adjusting for potential covariates.

Results: Neither PAH concentrations or obesity had a main effect on asthma or recent wheeze. In models stratified by presence/absence of obesity, a significant positive association was observed between an interquartile range (IQR) increase in natural log-transformed 1-methylphenanthrene (RR [95% CI]: 2.62 [1.17–5.88] with $IQR_{ln}=0.76$), and 9-methylphenanthrene (2.92 [1.09–7.82] with $IQR_{ln}=0.73$) concentrations and asthma in obese children ($n=63$). No association in non-obese ($n=248$) children was observed at age 5 ($P_{interaction}<0.03$). Similar associations were observed for 3-methylphenanthrene, 9-methylphenanthrene, and 3,6-dimethylphenanthrene at age 7.

Conclusions: Obese young children may be more likely to develop asthma in association with greater exposure to PAHs, and methylphenanthrenes in particular, than non-obese children.

Abbreviations: ACQ, Asthma Control Questionnaire; BMI, body mass index; BRQ, Brief Respiratory Questionnaire; BC, black carbon; CCCEH, Columbia Center for Children's Environmental Health; DEP, Diesel Exhaust Particle; ER, Emergency Room; EC, elemental carbon; ETS, environmental tobacco smoke; FEV_1, forced expiratory volume in 1 s; ICS, inhaled corticosteroids; IQR, interquartile range; ISAAC, International Study of Asthma and Allergies in Childhood; NO_2, nitrogen dioxide; NYC, New York City; PAH, polycyclic aromatic hydrocarbons; $\sum_8 PAH_{semivolatile}$, sum of 8 low molecular-weight-PAH ≤ 206, including phenanthrene (Phe), 1-methylphenanthrene (1Meph); 2-methylphenanthrene (2Meph), 3-methylphenanthrene (3Meph), 9-methylphenanthrene (9Meph), 1,7-dimethylphenanthrene (1,7DMeph), 3,6-dimethylphenanthrene (3,6DMeph), and pyrene; $\sum_8 PAH_{nonvolatile}$, sum of 8 high molecular-weight-PAH ≥ 228, including benz[a]anthracene (BaA), chrysene/iso-chrysene (Chry); benzo[b]fluoranthene (BbFA), benzo[k]fluoranthene (BkFA), benzo[a]pyrene (BaP), indeno[1,2,3-c,d]pyrene (IP), dibenz[a,h]anthracene (DahA), and benzo[g,h,i]perylene (BghiP); PM, particulate matter; PUF, Polyurethane Foam; RR, relative risk

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1. Introduction

The prevalence of childhood obesity has increased from 7% to 20% from 1980–2008 in the United States, and is higher for Hispanics (31%) and Blacks (23%) in New York City (NYC) (Thorpe et al., 2004). Concomitant with the rapid rise in childhood obesity, the prevalence of childhood asthma also has increased from 3.6% to 9.6% during 1980–2009 (Akinbami et al., 2009; CDC, 2011), especially in urban areas like NYC (Nicholas et al., 2005).

Exposure to air pollution, and polycyclic aromatic hydrocarbons (PAHs) in particular, appears to be a risk factor for the development of asthma in urban settings (Jung et al., 2012b; Miller et al., 2004). For example, our previous study reported that repeated high exposure to PAHs, semivolatile pyrene specifically, during prenatal period and age 5–6 years, is associated with asthma in young children (Jung et al., 2012b). Obesity also has been associated with the development of childhood asthma in both cross-sectional and prospective studies (Castro-Rodriguez et al., 2001; Gilliland et al., 2003; Visness et al., 2010). Further, the asthma-obesity association may vary by sex or atopic status (Castro-Rodriguez et al., 2001; Gilliland et al., 2003; Visness et al., 2010). For example, a study in the Tucson, Arizona cohort found that becoming overweight or obese between the ages of 6 and 11 years increased the risk of developing new wheeze at age 11 or 13 among girls but not boys (Castro-Rodriguez et al., 2001). However, a subsequent prospective study of school-age children in South California found that being overweight or obese was associated with an increased risk of incident asthma in boys, but not in girls; the strongest effect was observed among nonatopic boys (Gilliland et al., 2003). One study using the National Health and Nutrition Examination Survey (NHANES) data set (1999–2006) also found that obesity was associated with current asthma among children ages 2–19, with a stronger association in nonatopic children (Visness et al., 2010).

Emerging evidence suggests that the presence of obesity may modify the effects of exposure to air pollution on respiratory disease. For example, obese individuals experienced a greater decline in forced expiratory volume in 1 s (FEV_1) following ozone (O_3) exposure, compared to nonobese subjects (Alexeeff et al., 2007). This finding was supported by animal studies showing that genetically (db/db mice lacking leptin receptor) or diet-induced obese mice exhibited increased airway hyperresponsiveness (AHR) and enhanced O_3-induced pulmonary inflammation (e.g., greater interleukin (IL)-6 production) compared to nonobese mice (Johnston et al., 2008; Lu et al., 2006). These results suggest that obesity may confer greater vulnerability in airways following exposure to some air pollutants, yet the role of obesity following exposure to PAH on childhood asthma has not been studied previously.

PAHs have 2 types of anthropogenic sources: pyrogenic (i.e., incomplete combustion of organic material such as from traffic emissions and heating oil combustion) and petrogenic (e.g., unburned fossil organic material contribution such as direct evaporations from petroleum products). Alkylated PAHs (e.g., methylphenanthrenes), as opposed to parent PAHs (e.g., phenanthrene), are emitted more abundantly from petrogenic than pyrogenic sources (Saha et al., 2009). The toxicities of the individual phenanthrenes and methylaphenanthrenes during in vivo experiments are known to differ (Wolinska et al., 2011). While our earlier study suggests that repeated high exposure to unsubstituted semivolatile PAHs (i.e., pyrene) may induce asthma (Jung et al., 2012b), our understanding of the effects of exposure to alkylated PAHs (e.g., methylphenanthrenes) on respiratory health is more limited.

The goal of this study was to examine whether obesity may modify the effects of age 5–6 years PAH exposure, and semivolatile and alkylated PAHs in particular, on asthma in 5–7 years old inner-city children living in Northern Manhattan and the Bronx. Under the auspices of the Columbia Center for Children's Environmental Health (CCCEH) birth cohort, we were able to control for prenatal PAH concentrations and exposure to other indoor air pollutants such as fine particulate matter ($PM_{2.5}$) and soot/black carbon (BC). We hypothesized that obese children would be at greater risk of having asthma and recent wheeze with exposure to higher PAH than would non-obese children.

2. Methods

2.1. Study population

727 healthy and nonsmoking Dominican or African American mothers who reside in Northern Manhattan or South Bronx were enrolled into the CCCEH cohort during pregnancy (Miller et al., 2004). Questionnaires were administrated to the mother prenatally, every 3 months through age 2 years, and every 6 months thereafter. PAH concentrations were measured from residential indoor monitors for 408 of their children at age 5–6 years. Data were analyzed for those children ($n=363$) for whom measures of indoor PAH measures at age 5–6, height and weight, and complete age 5 questionnaire data were available. The study was approved by the Columbia University Institutional Review Board and written informed consent was obtained from all study participants.

2.2. Air monitoring

Details of residential air monitoring have been published previously (Jung et al., 2010a). Indoor air monitors were placed in a room where the child spent most of his or her time for two weeks between 2005 and 2011 at age 5 through 6. Sixteen 3-ring to 6-ring PAH (eight semivolatile ($178 \leq MW \leq 206$) and eight nonvolatile ($228 \leq MW \leq 278$) PAH; see Supplementary material) were selected as target compounds due to their abundance in traffic emissions and their possible carcinogenicity and mutagenicity (Jung et al., 2010b). Nine of these PAHs (eight nonvolatile PAHs and pyrene) also had been measured prenatally using 48-hr personal air monitoring between 1998 and 2006 (Miller et al., 2004). A single soxhlet extraction of both the filter and PUF together was analyzed for PAHs at Southwest Research Institute as described (Jung et al., 2010a, 2010b; Miller et al., 2004).

Residential indoor levels of $PM_{2.5}$ and BC were measured simultaneously with PAH monitoring, with a repeat sampling 6 months later. Details of analytical protocols for $PM_{2.5}$ and BC are described in Jung et al. (2010a, 2010b).

2.3. Obesity

Child's height and weight were measured at age 5, using a SECA wall-mounted stadiometer (SECA, Hamburg, Germany) and a Detecto Cardinal 750 digital scale (Cardinal Scale Manufacturing Company, Webby City, Missouri), respectively (Rundle et al., 2012). Children's body mass index (BMI; defined as weight in kilograms divided by height in meters squared (kg/m^2)) z scores and percentiles were calculated using the Centers for Disease Control (CDC) and Prevention SAS macro (Prevention, 2004). A child was classified as obese if the BMI was greater than or equal to the age- and sex-specific 95th percentile of the year 2000 CDC growth charts.

2.4. Health outcomes

The Brief Respiratory Questionnaires (BRQ) (Bonner et al., 2006) was used to assess current asthma, defined as parental report of doctor diagnosis of ever asthma AND a report of asthma medication use in the past 12 months. International Study of Asthma and Allergies in Childhood questionnaires (ISAAC) (Jenkins et al., 1996) was used to assess recent wheeze, defined as any report of wheeze in the last 12 months.

2.5. Seroatopy

Total and specific immunoglobulin (Ig)E to cat, mouse, dog, *Dermatophagoides farinae* and German cockroach at age 5 were measured in duplicate from sera using Immunocap (Phadia, Uppsala, Sweden) as described (Donohue et al., 2008). Children were considered sensitized to indoor allergens if they had a specific IgE $\geq$ 0.35 IU/mL to any of the allergens tested. They were classified as having any allergic sensitization if total IgE $\geq$ 50 IU/mL. Children were defined as seroatopic if they met either of these criteria.

2.6. Statistical analysis

Age 5–6 years PAH were natural log-transformed to normalize skewed distributions (e-Fig. 1). As reported previously (Jung et al., 2010a), PAH were analyzed as individual PAH, the sum of eight semivolatile PAH ($\Sigma_8 PAH_{semivolatile}$), and the sum of